Kaohsiung Journal of Medical Sciences (2015) 31, 83–89

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: http://www.kjms-online.com

ORIGINAL ARTICLE

Polysomnographic predictors of persistent continuous positive airway pressure adherence in patients with moderate and severe obstructive sleep apnea

Yung-Fu Chen [a], Liang-Wen Hang [b], Chun-Sen Huang [b], Shinn-Jye Liang [b], Wei-Sheng Chung [c,*]

[a] *Department of Healthcare Administration, Central Taiwan University of Science and Technology, Taichung, Taiwan*
[b] *Sleep Medicine Center, Department of Internal Medicine, China Medical University Hospital, Taichung, Taiwan*
[c] *Department of Internal Medicine, Taichung Hospital, Ministry of Health and Welfare, Taichung, Taiwan*

Received 7 April 2014; accepted 15 September 2014
Available online 18 December 2014

KEYWORDS
Adherence;
Continuous positive airway pressure (CPAP);
Obstructive sleep apnea (OSA);
Polysomnographic parameters

Abstract Extensive use of continuous positive airway pressure (CPAP) has positive clinical benefits for most patients with obstructive sleep apnea (OSA). However, patient adherence is a major limiting factor to the effectiveness of CPAP treatment. This study determined the potential and quantifiable factors affecting the willingness of patients with OSA to undertake CPAP treatment by comparing the polysomnographic parameters recorded during diagnosis and titration. Patients with moderate and severe OSA who attended diagnostic polysomnography (PSG) and CPAP titration at the sleep center of China Medical University Hospital (CMUH) were included in the study. A total of 312 patients were divided into persistent users and nonusers of CPAP according to their use of in-home CPAP following titration and a 7-day CPAP trial. Multivariate logistic regression analyses were used to define the potential polysomnographic predictors of persistent CPAP adherence, and odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. Most patients were men older than 50 years who were overweight or obese. Among the patients, 146 (46.8%) became persistent CPAP users. A 10% improvement of oxygen desaturation index (ODI) and a 10% increment in deep sleep percentage increased the chance of persistent CPAP use 1.18-fold and 1.07-fold, respectively. In addition, the improved ODI and

Conflicts of interest: All authors declare no conflicts of interest.

* Corresponding author. Department of Internal Medicine, Taichung Hospital, Department of Health, Executive Yuan 199, Section 1, San-Min Road, Taichung Taiwan.
E-mail address: chung.w53@msa.hinet.net (W.-S. Chung).

http://dx.doi.org/10.1016/j.kjms.2014.11.004

deep sleep during CPAP titration increased the chance of persistent CPAP user. The polysomnographic parameters obtained from diagnosis and during titration can facilitate the prediction of persistent CPAP use.

Introduction

Obstructive sleep apnea (OSA), a prevalent sleep disorder characterized by recurrent airway obstruction during sleep that causes hypoxemia and sleep fragmentation, can result in excessive daytime sleepiness [1], mood disturbance [2], deficits in neurobehavioral performance [3], deteriorated functional status and quality of life, increased risk of hypertension and cardiovascular disease, metabolic dysfunction, and traffic accidents [4—8]. Overweight and obesity are common predisposing factors for OSA [9], which frequently causes daytime sleepiness associated with impaired daytime performance and affects approximately 3—7% of middle-aged men and 2—5% of middle-aged women [10].

Two nights of polysomnography (PSG) is the typical length of time required to diagnose OSA and titrate continuous positive airway pressure (CPAP) [11] because breathing and CPAP responses during all sleep stages can adequately be observed. The use of CPAP is well established to effectively treat daytime manifestations of severe OSA. Extensive CPAP use affords substantial clinical benefits, such as reduced daytime sleepiness, systolic and diastolic blood pressure, insulin resistance, cardiovascular risk, oxidative stress, total cholesterol, and inflammation [12]. Evidence suggests that using CPAP for longer than 6 hours decreases sleepiness, improves daily functioning, and restores memory to normal levels [13].

However, poor CPAP compliance was observed in approximately 30% of patients with OSA in Taiwan [14], which is substantially lower than the poor CPAP compliance identified in Western countries (60—70%) [15]. Several studies have reported that complaints pertaining to CPAP use, such as inconvenience, poor mask fit, discomfort, skin irritation, mask leaks, sore eyes, dry airway, nasal complications, frequent awakening, claustrophobia, and aversion to CPAP treatment, might affect CPAP compliance [16,17]. Although several psychological and clinical parameters have been used to predict CPAP adherence, inconsistent results have been determined [17,18]. Various clinical predictors, including female sex, increased age, and reduced Epworth Sleepiness Scale (ESS) scores, have a considerable correlation with increased CPAP use [19,20]. In addition, the symptomatic severity of apnea is a critical predictor of long-term adherence [20].

Investigating the predictors of long-term CPAP adherence is crucial to treating OSA and lowering comorbidity risk. However, few identified variables can be used to reliably predict CPAP adherence. In this study, we determined the potential factors affecting the adherence of patients with OSA who had been advised to undergo CPAP treatment by using PSG parameters recorded during OSA diagnosis and CPAP titration.

Methods

Patients

We retrospectively reviewed the consecutive PSG records of patients with OSA who were treated at the sleep center of China Medical University Hospital (CMUH), Taichung, Taiwan between January 2007 and December 2009. The study was approved by the CMUH Medical Research Ethics Committee (DMR98-IRB-292). Fig. 1 shows that a total of 661 patients with OSA were assessed for eligibility in this study. After a 7-day trial, 349 patients were excluded because of poor compliance as a result of severe skin allergies, nasal congestion, sinusitis, stomach bloating, uncontrolled or severe bullous lung disease, pathologically low blood pressure, severe cardiac arrhythmia, uncontrolled coronary artery disease, stroke, seizure, or economic problems. The remaining 312 patients diagnosed with moderate [$15/h \le$ apnea/hypopnea index (AHI) $< 30/h$] and severe OSA (AHI $\ge 30/h$) according to the American Academy of Sleep Medicine (AASM) guidelines [21] and age $\ge$21 years [22,23] who underwent complete in-laboratory diagnostic PSG and CPAP titration were recruited for this study. CPAP titration was performed 1 month after the PSG diagnosis. Because the age range of pediatric patients began at the fetal stage and terminated at 21 years of age [22], only patients $\ge$21 years old were included in our investigation [23].

Patients were treated with CPAP using a fixed pressure determined by titration during a 7-day product trial at home prior to committing to purchase a CPAP device. To reduce bias, the CPAP devices (Type S8; Resmed, Martinsried, Germany) utilized during the titration and the home trial were manufactured by the same company. Patients were trained to use the CPAP and fitted for a mask by a certified sleep technician from the Taiwan Society of Sleep Medicine.

We assumed that patients who purchased a CPAP device were persistent users. Participants were categorized into persistent users and nonusers according to their use of in-home CPAP treatment. The economic status of patients substantially affected their willingness to purchase a CPAP device for OSA treatment; therefore, nonusers who did not purchase a device for economic reasons were excluded from further study. Demographic characteristics including sex, age, body mass index (BMI, kg/m^2), neck and waist circumferences (cm), and sleepiness index measured according to ESS scores graded on a 5-point Likert scale [24,25] were collected for analysis. CPAP titration was conducted by a certified sleep technician. An effective (optimal) CPAP pressure was determined during the one-night CPAP titration using the following procedure:

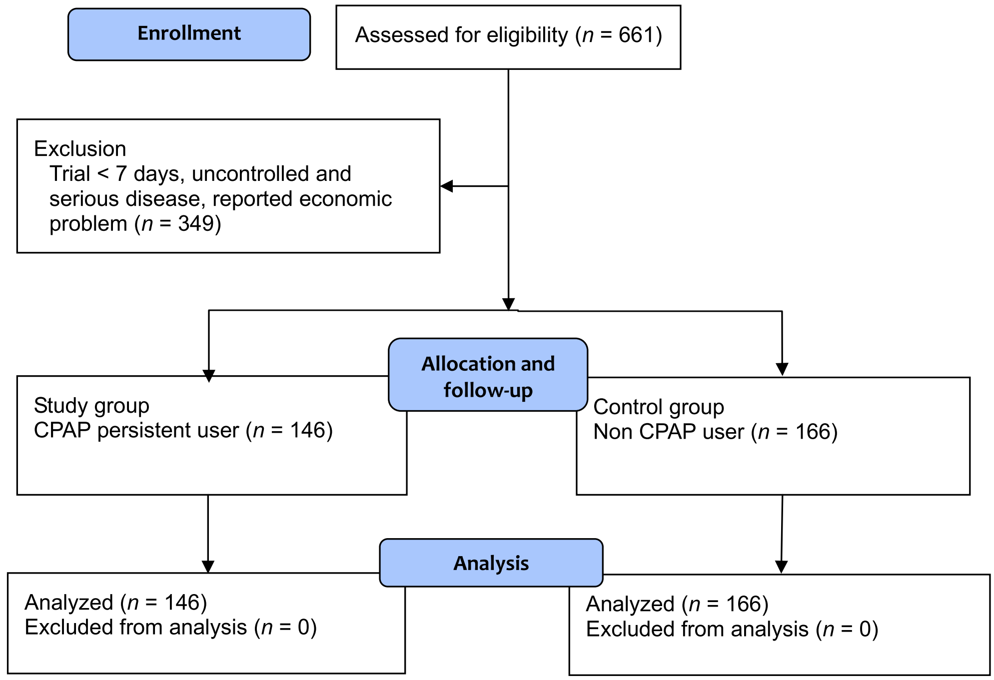

Figure 1. A flow diagram of the sampling scheme used in this study. CPAP = continuous positive airway pressure.

positive airway pressure was titrated upward in minimal increments of 1 cmH_2O in 5-minute intervals according to obstructive respiratory-related events (apneas, hypopneas, respiratory effort-related arousals, snoring). The titration procedure continued until no abnormal respiratory-related events occurred within 30 minutes [11].

PSG

Polysomnographic studies were conducted using the Sandmansleep sleep diagnostic system. Continuous recordings were used to monitor the following parameters: electroencephalogram, electrooculogram, electromyogram, electrocardiogram, respiratory effort, nasal pressure (measured and transmitted using a nasal cannula to a pressure transducer), oxyhemoglobin saturation (measured using a pulse oximeter), snoring duration (measured using a vibratory sensor), body position (measured using an accelerometer), and nasal flow (measured using a thermistor or thermocouple sensor to detect the temperature of air exhaled from the nose or mouth). Sleep stages and arousals were scored according to the criteria proposed by Rechtschaffen and Kales [26]. The spontaneous arousal index (AI) was defined as the number of spontaneous arousals multiplied by the number of hours of sleep. The oxygen desaturation index (ODI) was defined as the mean number of events per hour during which the oxygen saturation level decreased by $>3\%$ during sleep. An apnea was defined as a cessation of airflow for a minimum of 10 seconds; a hypopnea was determined by a 30% reduction in airflow for a minimum of 10 seconds and a peripheral capillary oxyhemoglobin desaturation (SpO_2) decrease of $\geq 4\%$ [27].

The apnea/hypopnea index (AHI) was calculated as the total number of apneas (central, mixed, or obstructive) and hypopneas per hour of sleep. The AI, the total number of arousals per hour of sleep, was determined according to respiratory effort-related episodes occurring with a minimum of 10 seconds of reduced airflow that did not fulfill apnea or hypopnea criteria and were terminated with arousal [27]. PSG recordings were performed overnight for a minimum of 6 hours when patients received OSA diagnoses and CPAP titration studies. Patients with recording periods that were <6 hours received another examination. Sleep efficiency was calculated as the ratio of the total sleep time (TST) to the total time spent in bed.

Working definitions of variables

Ten percent increments of deep sleep after CPAP titration: the difference during deep sleep (Stages III and IV called slow-wave sleep, SWS) between CPAP titration and initial diagnosis was measured using PSG in 10% increments.

Spontaneous AI decrease after CPAP titration: the decrease in spontaneous AI between CPAP titration and initial diagnosis, measured according to PSG.

Ten percent improvement in ODI after CPAP titration: the ODI difference between CPAP titration and initial diagnosis was measured to determine 10% improvement.

Statistical analysis

Statistical tests were performed using SPSS for Windows version 16 (SPSS Inc., Chicago, IL, USA). Descriptive statistics were used to describe the characteristics of the patients, and continuous and ordinal variables were presented as the mean $\pm$ standard deviation (SD). The Student *t* test was used to compare the means of baseline characteristics, polysomnographic data, CPAP titration, and differences between persistent users and nonusers of CPAP. Univariate logistic regression analysis was performed to identify the potential

demographic and polysomnographic variables predicting CPAP persistent users. Multivariate logistic regression analyses performed using a full model and a reduced model, including variables with $p < 0.05$ in the univariate analysis, were further used to define the predictors for persistent CPAP use. We did not include the AHI change variable in the multivariate logistic regression model because of a strong correlation to ODI. Odds ratios (ORs) and 95% confidence intervals (CIs) were also calculated.

Results

Baseline demographic and clinical characteristics of participants

Men accounted for 85.26% of the patients, and approximately 55% of the patients were older than 50 years. Most patients were overweight (53.85%, $24 \leq \text{BMI} \leq 30$ kg/m^2) or obese (30.77%, $\text{BMI} \geq 30$ kg/m^2). A total of 147 patients (47.11%) had a neck circumference exceeding 40 cm, and 244 patients (78.2%) had a waist circumference exceeding 90 cm. Among these patients, a substantial proportion were diagnosed with severe OSA (73.72%, $\text{AHI} \geq 30$/h). The distribution of patient ESS scores ranging from 0 to 5, 6 to 10, 11 to 15, 16 to 20, and >20 were 22.76%, 31.73%, 28.2%, 14.1%, and 3.2%, respectively. During the study, 146 patients (46.79%) became persistent users (Table 1).

Table 1 Baseline demographic and clinical characteristics of participants ($N = 312$).

Characteristics		No. of patients	%
Sex	Male	266	85.26
	Female	46	14.74
Age (y)	21–30	12	3.85
	31–40	44	14.10
	41–50	85	27.24
	51–60	89	28.53
	>60	82	26.28
BMI (kg/m^2)	BMI < 18.5	2	0.64
	18.5 ≤ BMI < 24	46	14.74
	24 ≤ BMI < 27	90	28.85
	27 ≤ BMI < 30	78	25.00
	30 ≤ BMI < 35	65	20.83
	BMI ≥ 35	31	9.94
Neck circumference (cm)	≤35.0	26	8.33
	35.1–40	139	44.55
	40.11–45.0	127	40.71
	45.1–50.0	18	5.77
	≥50	2	0.64
Waist circumference (cm)	≤80.0	17	5.45
	80.1–90.0	51	16.35
	90.1–100.0	134	42.95
	100.1–110.0	64	20.51
	≥100.0	46	14.74
ESS	≤5	71	22.76
	6–10	99	31.73
	11–15	88	28.2
	16–20	44	14.1
	≥20	10	3.21
AHI (/h)	Moderate (15.0–30.0)	82	26.28
	Severe (≥30)	230	73.72
Persistent CPAP users	Yes	146	46.79
	No	166	53.21

AHI = apnea/hypopnea index; BMI = body mass index; CPAP = continuous positive airway pressure; ESS = Epworth Sleepiness Scale.

Comparison of baseline characteristics between persistent users and nonusers

Table 2 indicates that persistent CPAP users had significantly greater BMIs (BMI: 29.08 ± 4.90 kg/m^2 vs. 27.22 ± 4.76 kg/m^2, $p = 0.017$), more severe OSA (AHI: 55.02 ± 25.44/h vs. 45.00 ± 23.59/h, $p < 0.001$), greater ODI (48.83 ± 28.92/h vs. 36.76 ± 23.75/h, $p < 0.001$), and a higher effective pressure during CPAP treatment (7.90 ± 2.96 cmH_2O vs. 7.05 ± 2.56 cmH_2O, $p = 0.006$) than the nonusers.

Comparison of polysomnographic parameters during the first night of OSA diagnosis between persistent users and nonusers

Persistently CPAP users had higher AHIs (55.02 ± 25.44/h vs. 45.00 ± 23.59/h, $p < 0.01$), larger AIs (46.15 ± 23.10/h vs. 37.64 ± 20.90/h, $p < 0.01$), greater ODIs (48.83 ± 28.92/h vs. 36.76 ± 23.75/h, $p < 0.01$), longer light sleep stages (83.00 ± 8.64% vs. 80.39 ± 8.88%, $p < 0.01$), shorter deep sleep stages (1.03 ± 2.54% vs. 2.05 ± 4.67%, $p < 0.05$), and a lower mean SpO_2 (91.79 ± 4.80% vs. 93.44 ± 2.68%, $p < 0.01$) and minimal SpO_2 (71.95 ± 11.36% vs. 75.61 ± 10.56%, $p < 0.01$) than nonusers (Table 3).

Changes in PSG parameters occurring between OSA diagnosis and CPAP titration in persistent users and nonusers

Persistent users had a considerably higher decrease in AHI (46.30 ± 22.95 vs. 36.97 ± 22.09), light sleep (8.92 ± 13.06 vs. 4.17 ± 10.63), spontaneous AI (18.86 ± 27.97 vs. 14.42 ± 24.25), and ODI (42.88 ± 26.40 vs. 31.15 ± 21.77) than nonusers. In addition, they had a higher increase in deep sleep percentage (1.20 ± 4.48% vs. -0.50 ± 4.56%) and rapid eye movement (REM) sleep percentage (7.73 ± 11.45% vs. 4.67 ± 9.18%) than nonusers (Table 4).

Clinical and polysomnographic predictors of persistent CPAP users

Demographic variables and changes in polysomnographic variables occurring between diagnosis and CPAP titration were considered to be predictors of persistent CPAP users. A 10% increment in deep sleep percentage and a 10% improvement in ODI percentage increased the chance of persistent CPAP use 1.07-fold and 1.18-fold, respectively, after controlling for the demographic variables and polysomnographic parameters.

Table 2 Comparison of baseline characteristics between CPAP persistent users and nonusers.

Variable	Persistent users ($N = 146$)	Nonusers ($N = 166$)	p*
	Mean ± SD	Mean ± SD	
Sex, male (%)	127 (87.0)	139 (83.7)	0.421
Mean age (y)	51.49 ± 12.96	53.67 ± 12.53	0.132
BMI (kg/m^2)	29.08 ± 4.90	27.22 ± 4.76	0.017
Neck circumference (cm)	40.52 ± 3.53	39.91 ± 3.51	0.126
Waist circumference (cm)	99.94 ± 12.48	97.34 ± 12.32	0.066
ESS score	10.6 ± 5.51	9.73 ± 5.16	0.155
AHI (/h)	55.02 ± 25.44	45.00 ± 23.59	<0.001
ODI (/h)	48.83 ± 28.92	36.76 ± 23.75	<0.001
Effective pressure of CPAP treatment (/h)	7.90 ± 2.96	7.05 ± 2.56	0.006

* Student t test.
AHI = apnea/hypopnea index; BMI = body mass index; CPAP = continuous positive airway pressure; ESS = Epworth Sleepiness Scale; ODI = oxygen desaturation index; SD = standard deviation.

Discussion

Frequently reported reasons for nonadherence to and discontinuation of CPAP therapy include embarrassment, physical discomfort, psychological factors (such as claustrophobia), and difficulty of use [13]. In previous studies that have defined adherence to CPAP usage as using CPAP for a minimum of 4 h/night, 29–83% of the patients were nonadherent [13]. In this study, we investigated the polysomnographic predictors of persistent CPAP use by excluding patients with financial problems and poor compliance. Men accounted for 85.26% of the recruited patients, a percentage that is higher than the 66.7% reported in a previous study [28]. The disparity between patient sex in our study and that in the previous study might have been because we enrolled participants who presented with moderate and severe OSA at a tertiary hospital. Persistent users of CPAP had more severe OSA than did nonusers ($p < 0.05$, Table 2), a phenomenon that was noted in previous studies reporting that patients with severe OSA and higher ESS scores were more likely to adhere to long-

Table 3 Comparison of polysomnographic parameters at diagnosis between CPAP persistent users and nonusers.

Variable	Persistent users ($N = 146$)	Nonusers ($N = 166$)	p*
	Mean ± SD	Mean ± SD	
TST (min)	301.51 ± 52.42	300.22 ± 54.07	0.832
Sleep efficiency (%)[a]	79.84 ± 14.05	78.57 ± 14.80	0.437
Sleep latency (min)	11.72 ± 13.54	15.16 ± 21.99	0.093
No. of hypopneas, Stage I	83.30 ± 39.30	74.66 ± 37.54	0.048
Stage I, % of TST	44.21 ± 25.16	38.77 ± 21.79	0.044
Stage II, % of TST	38.79 ± 21.50	41.62 ± 19.17	0.224
Stage I + II, % of TST	83.00 ± 8.64	80.39 ± 8.88	0.009
Stage III, % of TST	0.97 ± 2.35	1.67 ± 3.60	0.039
Stage IV, % of TST	0.06 ± 0.42	0.38 ± 1.82	0.029
Stage III + IV, % of TST	1.03 ± 2.54	2.05 ± 4.67	0.019
REM sleep, % of TST	15.97 ± 7.92	17.56 ± 7.82	0.075
AI (/h)	46.15 ± 23.10	37.64 ± 20.90	0.001
AHI (/h)	55.02 ± 25.44	45.00 ± 23.59	<0.001
ODI (/h)	48.83 ± 28.92	36.76 ± 23.75	<0.001
SpO_2, mean (%)	91.79 ± 4.80	93.44 ± 2.68	<0.001
SpO_2, minimum (%)	71.95 ± 11.36	75.61 ± 10.56	0.003

* Student t test.
AHI = apnea/hypopnea index; AI = arousal index; CPAP = continuous positive airway pressure; ODI = oxygen desaturation index; REM = rapid eye movement; SD = standard deviation; SpO_2 = peripheral capillary oxyhemoglobin desaturation; TST = total sleep time.
[a] Sleep efficiency (%) = (TST/total time in bed) × 100.

Table 4 Comparison of CPAP persistent users and nonusers regarding changes in PSG parameters between diagnosis and CPAP titration.

Variable	Persistent users ($N = 146$)	Nonusers ($N = 166$)	p*
Light sleep[a] % of TST	−8.92 ± 13.06	−4.17 ± 10.63	<0.001
Deep sleep[b] % of TST	1.20 ± 4.48	−0.50 ± 4.56	0.001
REM sleep % of TST	7.73 ± 11.45	4.67 ± 9.18	0.010
Spontaneous AI	−18.86 ± 27.97	−14.42 ± 24.25	<0.001
AHI	−46.30 ± 22.95	−36.97 ± 22.09	<0.001
ODI	−42.88 ± 26.40	−31.15 ± 21.77	<0.001
Sleep efficiency (%)	5.04 ± 14.00	3.20 ± 13.26	0.227

Data are presented as mean ± SD.
* Student t test.
AHI = apnea/hypopnea index; AI = arousal index; CPAP = continuous positive airway pressure; ODI = oxygen desaturation index; PSG = polysomnography; REM = rapid eye movement; SD = standard deviation; TST = total sleep time.
[a] Light sleep = Stages I + II.
[b] Deep sleep = Stages III + IV.

Table 5 Demographic and polysomnographic predictors of persistent CPAP users.

	Univariate	Multivariate	
		Full model	Reduced model**
Variable	OR (95% CI)	OR (95% CI)	OR (95% CI)
Sex	1.30 (0.69–2.45)		
Age (y)	0.99 (0.97–1.01)		
10% increment in BMI %	1.77 (1.10–2.84)*	1.19 (0.68–2.11)	
Neck circumference	1.05 (0.99–1.12)		
Sleepiness index (ESS score)	1.03 (0.99–1.08)		
Effective pressure of CPAP during titration	1.13 (1.03–1.23)**	1.03 (0.92–1.15)	
10% increment in deep sleep % after CPAP titration	1.10 (1.03–1.16)**	1.07 (1.00–1.14)*	1.07 (1.00–1.13)*
Spontaneous AI decrease after CPAP titration	1.02 (1.01–1.03)**	1.00 (0.98–1.02)	
AHI change	1.02 (1.01–1.03)***		
10% improvement in oxygen desaturation index after CPAP titration	1.22 (1.11–1.35)***	1.18 (1.00–1.41)	1.18 (1.07–1.31)**

Multivariate logistic regression analysis using a full model and a reduced model, including variables with $p < 0.05$ in the univariate analysis: *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.
AHI = apnea/hypopnea index; AI = arousal index; BMI = body mass index; CI = confidence interval; CPAP = continuous positive airway pressure; ESS = Epworth Sleepiness Scale; OR = odds ratio.

term CPAP treatment [12,29] and required a higher effective treatment pressure. Early intervention and education about treatment benefits, expectations, and common problems encountered when trying to sleep when using a CPAP device can facilitate establishing an effective CPAP treatment [30,31]. Short-term adherence can influence long-term patterns of CPAP adherence; sleep quality experienced during the first 3 months and even the first few days of CPAP therapy can enable predicting long-term use [31]. Age and one-time use of sedatives or hypnotics during PSG have been associated with greater short-term CPAP compliance [18].

A 10% increment in deep sleep percentage increased the change of persistent CPAP use 1.07-fold after demographic and polysomnographic variables were adjusted for ($p < 0.05$; Table 5). In addition, compared with nonusers, persistent users experienced less light sleep (Stages I and II) and more deep sleep (Stages III and IV; Table 4), indicating that improved sleep quality plays a critical role in CPAP adherence.

Because of a strong correlation to ODI [32,33], the variable AHI was removed during the multivariate logistic regression analysis. A 10% improvement in ODI after CPAP titration significantly predicted persistent CPAP use (OR = 1.18, 95% CI = 1.07–1.31) in a multivariate reduced model that adjusted for demographic variables and polysomnographic variables. Although they are the most effective type of PSG, the Level 1 PSG examinations used in OSA diagnosis and CPAP titration are costly, complex, and time-consuming. However, the Level 4 PSG portable recording device that can be used for in-home monitoring can be feasible because persistent CPAP users tend to have lower ODIs and better sleep quality during CPAP titration. Diagnosing OSA at home can reduce the possibility of CPAP-induced insomnia and is less expensive than examinations conducted at sleep centers [31,32,34,35].

Several limitations must be considered when interpreting these findings. First, this was a retrospective study. The results might be biased because of data collection from a tertiary medical center, such as the issues of sex and prevalence. Second, we defined persistent CPAP use as continuous use for 1 month after diagnosis. However, the real time of CPAP use was undeterminable because the CPAP devices were not equipped with timers. Third, because two nights of PSG was time-consuming for patients and resource-intensive for the sleep center, CPAP titration was typically arranged 1 month after diagnosis, and during this waiting period, the physical conditions of patients might have changed. Fourth, information pertaining to CPAP therapy follow-up was unavailable. In addition, long-term treatment efficacy and PSG parameter variations between laboratory and in-house use could not be determined. Lastly, because patients with poor compliance were excluded, we cannot ensure this exclusion did not affect adherence.

In conclusion, this study indicated that CPAP adherence was associated with longer deep sleep and improved ODI during CPAP titration. Improving sleep quality and reducing oxygen desaturation is critical if patients with OSA are to become persistent CPAP users.

Acknowledgments

This study was supported in part by the Ministry of Science and Technology of Taiwan under grant NSC98-2410-H-039-003-MY2.

References

[1] Black J. Sleepiness and residual sleepiness in adults with obstructive sleep apnea. Respir Physiol Neurobiol 2003;136:211–20.
[2] Ohayon MM. The effects of breathing-related sleep disorders on mood disturbances in the general population. J Clin Psychiatry 2003;64:1195–200. quiz 274–6.
[3] Lal C, Strange C, Bachman D. Neurocognitive impairment in obstructive sleep apnea. Chest 2012;141:1601–10.

[4] Marin JM, Agusti A, Villar I, Forner M, Nieto D, Carrizo SJ, et al. Association between treated and untreated obstructive sleep apnea and risk of hypertension. JAMA 2012;307:2169—76.
[5] Lurie A. Cardiovascular disorders associated with obstructive sleep apnea. Adv Cardiol 2011;46:197—266.
[6] Martinez-Garcia MA, Campos-Rodriguez F, Catalan-Serra P, Soler-Cataluna JJ, Almeida-Gonzalez C, De la Cruz Moron I, et al. Cardiovascular mortality in obstructive sleep apnea in the elderly: role of long-term continuous positive airway pressure treatment: a prospective observational study. Am J Respir Crit Care Med 2012;186:909—16.
[7] Ellen RL, Marshall SC, Palayew M, Molnar FJ, Wilson KG, Man-Son-Hing M. Systematic review of motor vehicle crash risk in persons with sleep apnea. J Clin Sleep Med 2006;2:193—200.
[8] Sharma SK, Agrawal S, Damodaran D, Screenivas V, Kadhiravan T, Lakshmy R, et al. CPAP for the metabolic syndrome in patients with obstructive sleep apnea. N Engl J Med 2011;365:2277—86.
[9] Romero-Corral A, Caples SM, Lopez-Jimenez F, Somers VK. Interactions between obesity and obstructive sleep apnea: implications for treatment. Chest 2010;137:711—9.
[10] Jennum P, Riha RL. Epidemiology of sleep apnoea/hypopnoea syndrome and sleep-disordered breathing. Eur Respir J 2009;33:907—14.
[11] Kushida CA, Chediak A, Berry RB, Brown LK, Gozal D, Iber C, et al. Clinical guidelines for the manual titration of positive airway pressure in patients with obstructive sleep apnea. J Clin Sleep Med 2008;4:157—71.
[12] McDaid C, Duree KH, Griffin SC, Weatherly HL, Stradling JR, Davies RJ, et al. A systematic review of continuous positive airway pressure for obstructive sleep apnoea-hypopnoea syndrome. Sleep Med Rev 2009;13:427—36.
[13] Weaver TE, Grunstein RR. Adherence to continuous positive airway pressure therapy: the challenge to effective treatment. Proc Am Thorac Soc 2008;5:173—8.
[14] Yang MC, Lin CY, Lan CC, Huang CY, Huang YC, Lim CS, et al. Factors affecting CPAP acceptance in elderly patients with obstructive sleep apnea in Taiwan. Respir Care 2013;58:1504—13.
[15] Verse T, Pirsig W, Stuck BA, Hormann K, Maurer JT. Recent developments in the treatment of obstructive sleep apnea. Am J Respir Med 2003;2:157—68.
[16] Engleman HM, Wild MR. Improving CPAP use by patients with the sleep apnoea/hypopnoea syndrome (SAHS). Sleep Med Rev 2003;7:81—99.
[17] Stepnowsky Jr CJ, Marler MR, Ancoli-Israel S. Determinants of nasal CPAP compliance. Sleep Med 2002;3:239—47.
[18] Collen J, Lettieri C, Kelly W, Roop S. Clinical and polysomnographic predictors of short-term continuous positive airway pressure compliance. Chest 2009;135:704—9.
[19] Engleman HM, Martin SE, Douglas NJ. Compliance with CPAP therapy in patients with the sleep apnoea/hypopnoea syndrome. Thorax 1994;49:263—6.
[20] Sin DD, Mayers I, Man GC, Pawluk L. Long-term compliance rates to continuous positive airway pressure in obstructive sleep apnea: a population-based study. Chest 2002;121:430—5.
[21] Epstein LJ, Kristo D, Strollo Jr PJ, Friedman N, Malhotra A, Patil SP, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med 2009;5:263—76.
[22] American Academy of Pediatrics Council on Child and Adolescent Health. Age limits of pediatrics. Pediatrics 1988;81:736.
[23] Moser D, Anderer P, Gruber G, Parapatics S, Loretz E, Boeck M, et al. Sleep classification according to AASM and Rechtschaffen & Kales: effects on sleep scoring parameters. Sleep 2009;32:139—49.
[24] Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep 1991;14:540—5.
[25] Johns MW. Daytime sleepiness, snoring, and obstructive sleep apnea. The Epworth Sleepiness Scale. Chest 1993;103:30—6.
[26] Rechtschaffen A, Kales A. A manual of standardized terminology, technique and scoring systems for sleep stages of human subjects. Los Angeles: Brain Information Service/Brain Research Institute, University of California; 1968.
[27] Iber C, Ancoli-Israel S, Chesson A, Quan SF. The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications. Westchester, IL: American Academy of Sleep Medicine (AASM); 2007.
[28] Chen YH, Keller JK, Kang JH, Hsieh HJ, Lin HC. Obstructive sleep apnea and the subsequent risk of depressive disorder: a population-based follow-up study. J Clin Sleep Med 2013;9:417—23.
[29] Yetkin O, Kunter E, Gunen H. CPAP compliance in patients with obstructive sleep apnea syndrome. Sleep Breath 2008;12:365—7.
[30] Aloia MS, Arnedt JT, Stanchina M, Millman RP. How early in treatment is PAP adherence established? Revisiting night-to-night variability. Behav Sleep Med 2007;5:229—40.
[31] Damjanovic D, Fluck A, Bremer H, Muller-Quernheim J, Idzko M, Sorichter S. Compliance in sleep apnoea therapy: influence of home care support and pressure mode. Eur Respir J 2009;33:804—11.
[32] Torre-Bouscoulet L, Castorena-Maldonado A, Banos-Flores R, Vazquez-Garcia JC, Meza-Vargas MS, Perez-Padilla R. Agreement between oxygen desaturation index and apnea-hypopnea index in adults with suspected obstructive sleep apnea at an altitude of 2240 m. Arch Bronconeumol 2007;43:649—54.
[33] Marcos JV, Hornero R, Alvarez D, Aboy M, Del Campo F. Automated prediction of the apnea-hypopnea index from nocturnal oximetry recordings. IEEE Trans Biomed Eng 2012;59:141—9.
[34] Chung F, Liao P, Elsaid H, Islam S, Shapiro CM, Sun Y. Oxygen desaturation index from nocturnal oximetry: a sensitive and specific tool to detect sleep-disordered breathing in surgical patients. Anesth Analg 2012;114:993—1000.
[35] Chang L, Wu J, Cao L. Combination of symptoms and oxygen desaturation index in predicting childhood obstructive sleep apnea. Int J Pediatr Otorhinolaryngol 2013;77:365—71.